The effect of inorganic salts on renal tissue dopamine levels in the rat

S.G. BALL, M.R. LEE &
N.S. OATES

Department of Pharmacology and Department of Medicine, University of Leeds.

Increasing dietary sodium chloride in the rat results in an increased urinary excretion of free dopamine (Ball & Lee, 1977a). Further work has demonstrated that the administration of the chlorides of potassium and ammonium also raises the urinary free dopamine, whereas giving sodium bicarbonate results in a fall (Ball, Oates & Lee, unpublished observations). The urinary changes in free dopamine (a weak base) could be influenced by the pH of the urine. We have, therefore, examined the changes in free dopamine in the kidney, brain and liver of rats given the chlorides of sodium, potassium and ammonium; and sodium bicarbonate.

Male Wistar rats (200–250 g) housed in groups of six were given either a low sodium diet (BP Nutrition ref. 821511) or the same food supplemented by 0.85 mmol of sodium, potassium or ammonium, as their chlorides, per g of food; or 0.85 mmol of sodium bicarbonate per g of food. Both the control and test groups of rats received the diet for four days, after which they were killed.

Kidney, liver and brain from each rat were homogenized in 5 volumes of ice-cold 0.155 M KCl. After deproteinization with perchloric acid the tissue-free dopamine was measured by the radiokinetic method of da Prada & Zürcher (1976). The recovery of dopamine added to tissue homogenates by this method was 55.3 ± 3.2% (mean ± s.e. mean).

Kidney tissue dopamine in the NaCl group was 3.51 ± 0.10 pmol/mg protein (mean ± s.e. mean) compared with its low sodium control group of 3.06 ± 0.12 pmol/mg protein ($t = 2.98$; $P < 0.02$); in the KCl group was 3.56 ± 0.08 pmol/mg protein compared with its control of 3.22 ± 0.08 pmol/mg protein ($t = 2.80$; $P < 0.02$); in the NH_4Cl group was 3.33 ± 0.20 pmol/mg protein compared with its control of 2.78 ± 0.11 pmol/mg protein ($t = 2.84$; $P < 0.02$). There was no change in the $NaHCO_3$ group (3.73 ± 0.12 pmol/mg protein) compared with its control of 3.63 ± 0.11 pmol/mg protein ($t = 0.61$; $P > 0.1$). No significant changes in the liver or brain free dopamine were found in any of the groups of rats ($P > 0.1$).

The changes in renal dopamine reflect the changes we have found in the urinary dopamine of rats treated similarly. The absence of a change in hepatic or cerebral dopamine levels suggests that the increments in renal dopamine are specific to this tissue. The simple hypothesis that intrarenal free dopamine is a factor in the control of sodium excretion (Ball & Lee, 1977b; Faucheaux, Buu & Kuchel, 1977) must now take account of the effects of potassium and ammonium chlorides, and sodium bicarbonate. The changes in intrarenal dopamine may represent an integrated response to the combined stimuli of osmolarity and hydrogen ion concentration.

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The effects of some neurotransmitter substances on the production of corticotrophin releasing factor by the rat hypothalamus *in vitro*

JULIA C. BUCKINGHAM &
J.R. HODGES

Department of Pharmacology, Royal Free Hospital School of Medicine, 8 Hunter Street, London WC1N 1BP.

The hypothalamus of the rat is viable *in vitro* (Bradbury, Burden, Hillhouse & Jones, 1974) and responds to certain neurotransmitter substances with changes in the level of its functional activity. We have used this preparation to study the production of corticotrophin releasing factor (CRF). Hypothalami were removed from rats and incubated as previously described by Buckingham & Hodges (1977a) in the presence and absence of various neurotransmitter substances and drugs which mimic or antagonize their actions. The CRF content of the hypothalami and the media in which they were incubated were determined using the sensitive and precise method which depends on the ability of the hypothalamic hormone to stimulate corticotrophin production by